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COMPANY PRESENTATION

March 2020

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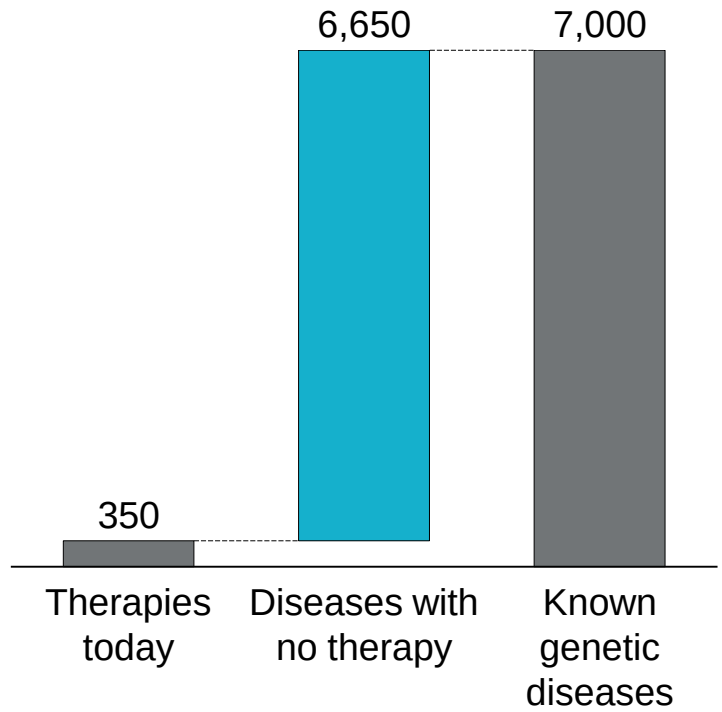
We are at Day 1 in the era of genetic medicine

Advances in science and medicine (2019)

- ***Better context***: Cryptic genetic variation and modifiers
- ***Better understanding heterogeneity***: Genetic interaction manifolds and the wonderful story of Hirschsprung's Disease
- ***Deeper saturation***: Saturation genome editing
- ***Faster***: Rapid whole-genome sequencing in the ICU
- ***Developing infrastructure***: National Human Genome Research Institute (NHGRI) reports cost per genome at $942 this year (all time low)
- ***Striking new therapeutics***: SCD, CF, PN, TTR, SMA, and others

Vast opportunity to help patients



Source: Science, Science Translational Medicine, Nature, NEJM, NHGRI

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We are building a leading genetic disease company

Core attributes…

1. **Distinctive early stage asset selection**
2. **Experienced, product-focused R&D team**
3. **Efficient corporate structure**
4. **The willingness and scale to fail**
5. **Focus at the level of individual diseases and assets**

…applied many times…

eidos a bridgebio company

pellepharm a bridgebio company

MYOKARDIA

GBT

+ 18 BridgeBio programs

…a pipeline of potential blockbusters and synthetic blockbusters*

- **Two potential $1B+ franchises in Phase 2 or later**
- **Two planned NDA submissions this year**
- **Several early-stage potentially large franchises**
  - KRAS
  - GPX4
  - Congenital adrenal hyperplasia
  - Leber's hereditary optic neuropathy
- **Multiple IND submissions planned in 2020**
- **Four new programs announced in January 2020**

*Blockbuster defined as program with $1bn+ opportunity

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BridgeBio is led by a world-class team of experienced drug developers

We rely on some of the top R&D minds in this industry to select assets…

Charles Homcy, MD Chairman of Pharmaceuticals	PORTOLA PHARMACEUTICALS, MyoKardia, globalblood THERAPEUTICS, Takeda MILLENNIUM THE TAKEDA ONCOLOGY COMPANY	VELCADE (bortezomib) FOR INJECTION, Andexxa (Coagulation Factor Xa (Recombinant), inactivated - zhzo), Oxbryta (voxelotor)
Frank McCormick, PhD, FRS Chairman of Oncology	Cetus, ONYX PHARMACEUTICALS, UCSF	Nexavar (sorafenib) tablets, Kyprolis (carfilzomib) for injection
Richard Scheller, PhD Chairman of R&D	Genentech, 23andMe	TECENTRIQ atezolizumab, PERJETA pertuzumab for injection, Kadcyla trastuzumab emtansine, OCREVUS ocrelizumab, VENCLEXTA venetoclax tablets

…and put them in the hands of one of the most productive groups of R&D operators in the industry

Uma Sinha, PhD Chief Scientific Officer	globalblood THERAPEUTICS, PORTOLA PHARMACEUTICALS, Takeda MILLENNIUM THE TAKEDA ONCOLOGY COMPANY	(eptifibatide) Injection INTEGRILIN, Andexxa (Coagulation Factor Xa (Recombinant), inactivated - zhzo), Oxbryta (voxelotor)
Eli Wallace, PhD Chief Scientific Officer in Residence, Oncology	ARRAY BIOPHARMA, Peloton Therapeutics	MEKTOVI (binimetinib) 15 mg tablets, Tucatinib (HER2i, Ph3), PT2997 (HIF2αi, Ph3)
Robert Zamboni, PhD Chemistry	MERCK FROSST	SINGULAIR (montelukast, MSD), ARCOXIA (etoricoxib), VIOXX (rofecoxib, MSD)

Together, our R&D team is responsible for 100+ INDs and 20+ approved products

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Assessing BridgeBio

	Criteria	Relevance	Focus Today
1	High probability of success	• Historically higher probability of success for genetic disease drugs • BridgeBio's early programs have outperformed historical probabilities	Current Pipeline Progress
2	Number of programs	• We find great science and unlock its potential for patients • Always searching for the next PellePharm or Eidos • Scale allows for objective assessment and failure	New Programs
3	Capital efficiency	• Generate value by making programs ROI-positive • Driven by judicious use of capital at the high-risk preclinical stages	Spend to IND

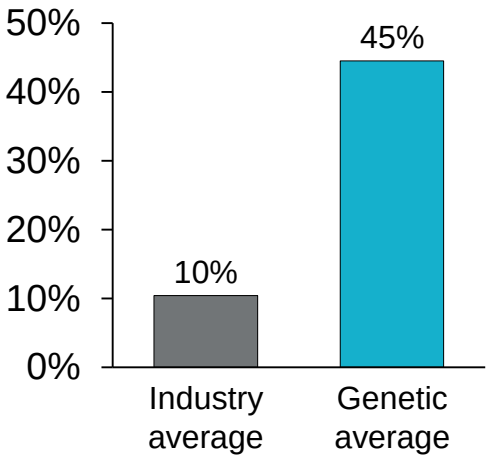
We believe genetic disease drug discovery is lower risk, faster, with potentially higher returns than traditional drug discovery

>4x Higher cumulative probability of success

>65% Faster time to Phase 3*

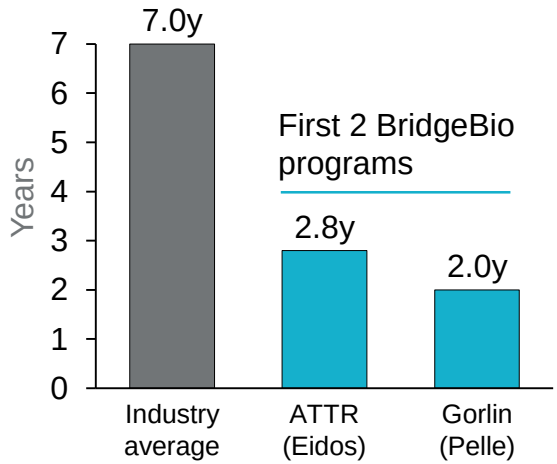
>8x Better return on investment*

Probability of success from Ph1 to launch



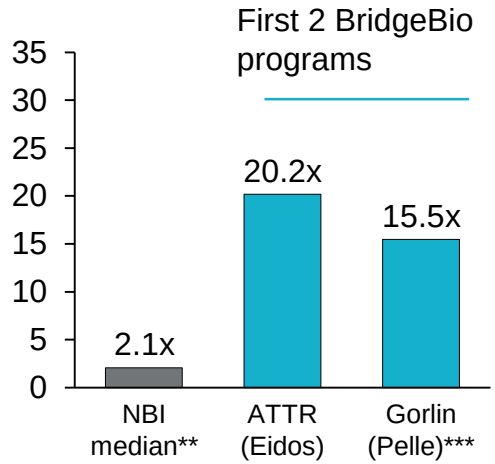
Sources: Hay et al., Nature Biotechnology, "Clinical Development Success Rates for Investigational Drugs", 2014

Time from lead optimization to Ph3



Sources: Paul et al., Nat Rev Drug Disc, "How to improve R&D productivity: the pharmaceutical industry's grand challenge.", 2010

Total return on investment
[Enerprise value]/[APIC - cash on hand]*



*As of 2/28/2020 close
**Includes all NBI constituents with market value <$20bn
***Calculated as total consideration from LEO Pharma transaction divided by total burn to date
Sources: FactSet

Targeting genetic disease has higher average probability of success and BridgeBio has demonstrated higher ROI and shorter development time in its first 2 programs

*For first two BridgeBio programs

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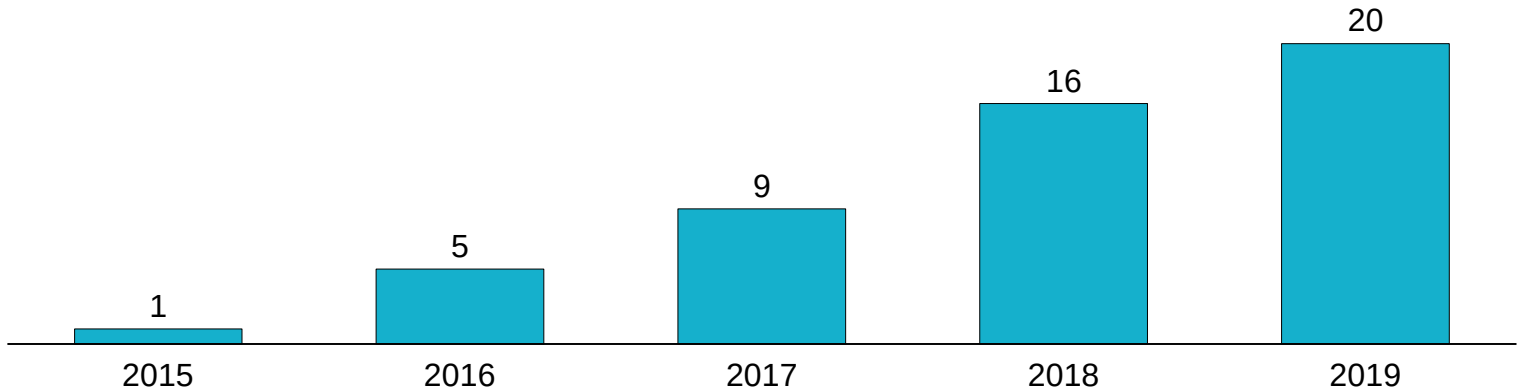
A rapidly-advancing pipeline

Since our inception, we have actively built our pipeline through business development efforts, including the acquisition and in-licensing of assets, and advancing programs through internal stage-gates

Growth of assets in our pipeline:

Total programs



Advancement of product candidates through key stage-gates:

Internal program advancement

	Lead-Opt	IND enabling	Ph1	Ph2	Ph3
2016	→	+1			
2017		→	+1		
2018	→	+1	→	+1	
2019	→	+6	→ +1	→	+3

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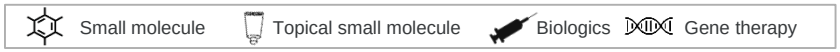
Our pipeline of 20+ development programs spans multiple therapeutic areas and drug modalities



Portfolio segment	Program[1]	Drug mechanism	Diseases	Patient pop. (US+EU)[2]	Modality
Mendelian	**AG10**	TTR stabilizer	ATTR-CM	>400K	
	BBP-870	cPMP replacement	MoCD type A	100	
	Infigratinib	Low-dose FGFR1-3i	Achondroplasia[4]	55K	
New program	**Encaleret**	CaSR antagonist	ADH1 / HP	2K / 200K	
New program	**Zuretinol**	Synthetic retinoid	IRD (RPE65 or LRAT)	3K	
	BBP-418	Glycosylation substrate	LGMD2i	7K	
	BBP-711	GO1 inhibitor	PH1 / FSF	5K / 1.5M	
	BBP-761	Succinate prodrug	LHON	20K	
	BBP-671	PanK activator	PKAN / OA	7K	
New program	**BBP-472**	PI3Kβi	PTEN autism	120K	
Genetic Dermatology	**Patidegib[3]**	Topical SMOi	Gorlin / BCC	120K	
	BBP-589	Recombinant COL7	RDEB	1.5K	
	BBP-681	Topical PI3Kαi	VM / LM	117K	
	BBP-561	Topical KLK 5/7i	Netherton	11K	
Targeted Oncology	**Infigratinib**	FGFR1-3i	FGFR+ tumors	37K	
	BBP-398	SHP2i	Multiple tumors	>500K	
	BBP-454	Pan-mutant KRASi	KRAS+ tumors	>500K	
	BBP-954	GPX4i	Multiple tumors	>500K	
Gene Therapy	**BBP-631**	21-OH gene therapy	CAH	>75K	
	BBP-812	ASPA gene therapy	Canavan	1K	
New program	**BBP-815**	TMC1 gene therapy	Genetic hearing loss	10K	

[1] Each of our programs is housed in a separate subsidiary; [2] Patient population: Prevalence except for asterisked figures which represent incidence; [3]We are party to an option agreement pursuant to which LEO Pharma A/S has been granted an exclusive, irrevocable option to acquire PellePharm, including the BBP-009 program. If the option is exercised by LEO Pharma A/S, we will no longer have rights to develop and commercialize BBP-009. [4]Protocol accepted by Australian local ethics committed, IND submission to FDA expected 2020.

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Low-dose FGFR inhibitor (infigratinib) for achondroplasia

Claudia, child with achondroplasia

Achondroplasia overview:

- **Prevalence**: 55,000 (US+EU) – one of the most common genetic conditions
- **Genetic driver**: FGFR3 activation
- **Pathophysiology**: Up regulation of STAT1 and MAPK in the growth plate cause cranial, spinal, and stature symptoms

Features of a potential best-in-class medicine for achondroplasia:

- **Direct targeting of FGFR3** and normalization both STAT1 and MAPK signaling pathways
- **Potential to address all drivers of symptoms**, including cranial, spinal and stature issues
- **Oral dosing**, the most convenient solution for children with achondroplasia and their families

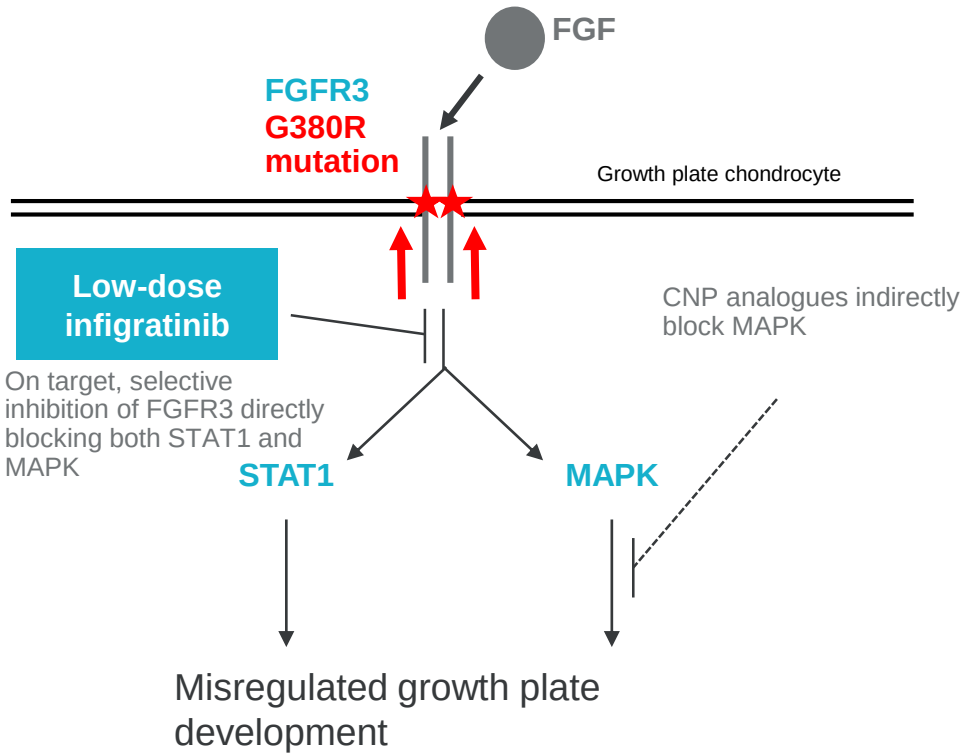
Potential best-in-class approach to treating achondroplasia directly at its genetic source

ACH FGFR3 gain-of-function mutation causes:

- 2-3x over activation of the receptor
- Up-regulation of downstream pathways STAT1 and MAPK
- Aberrant growth plate development causes cranial, spinal, and stature symptoms

Low-dose infigratinib has the potential to:

- Directly inhibit the causal gain-of-function mutation in FGFR3
- Normalize both the STAT1 and MAPK signaling pathways
- Demonstrate clear macro and microscopic improvements on foramen magnum, intervertebral discs, and long bones in validated preclinical model



Source: Ornitz DM et al. Developmental Dynamic 2017; Richette Joint Bone Spine 2007; Unger Curr Osteoporos Rep 2017, Hoover-Fong Am J Gen Med 2017

Low-dose infigratinib improves all the key drivers of clinical symptomology in validated ACH mouse model

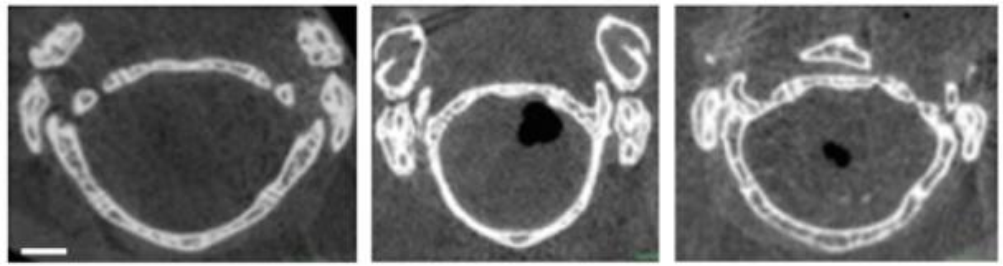
1 Cranial bone issues

17% increase in FM area

6% increase in AP skull length

May lead to **decrease** in **foramen magnum stenosis** and fewer surgeries

FGFR3 WT No treatment | **FGFR3$^{Y367C/+}$** No treatment | **FGFR3$^{Y367C/+}$** **Infigratinib tx**

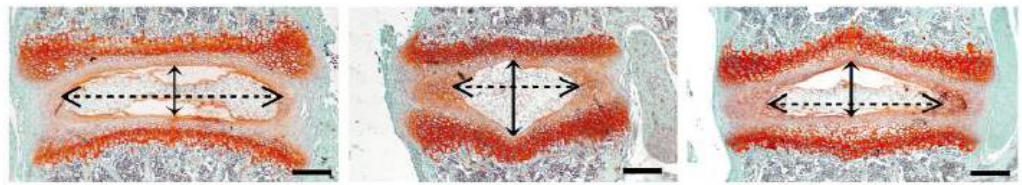


2 Disorders of the spine

12% increase in L4-L6 length

73% increase in disc width

May lead to **decrease** in **spinal stenosis**, possibly **reducing need for surgery**

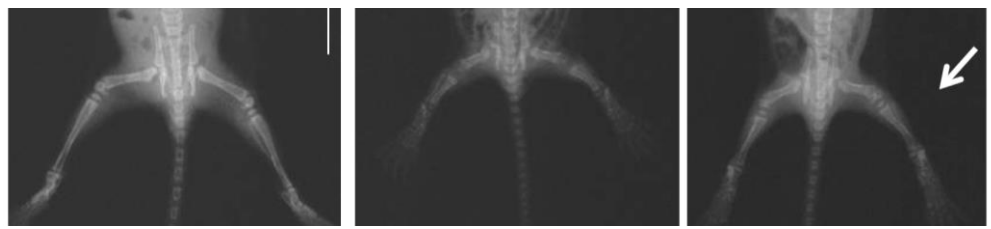


3 Disproportionate short stature

21% increase in femur length

33% increase in tibia length

May lead to **increased stature** and **proportionality**



Source: Komla-Ebri et al. J Clin Inv 2016

Note: percent increase compared to vehicle treated FGFR3$^{Y367C/+}$ mouse, infigratinib treatment with 2mg/kg subcutaneous dose

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Low-dose infigratinib showed potential best in-class preclinical profile in validated achondroplasia mouse model

Preclinical data from infigratinib and other investigational achondroplasia therapies

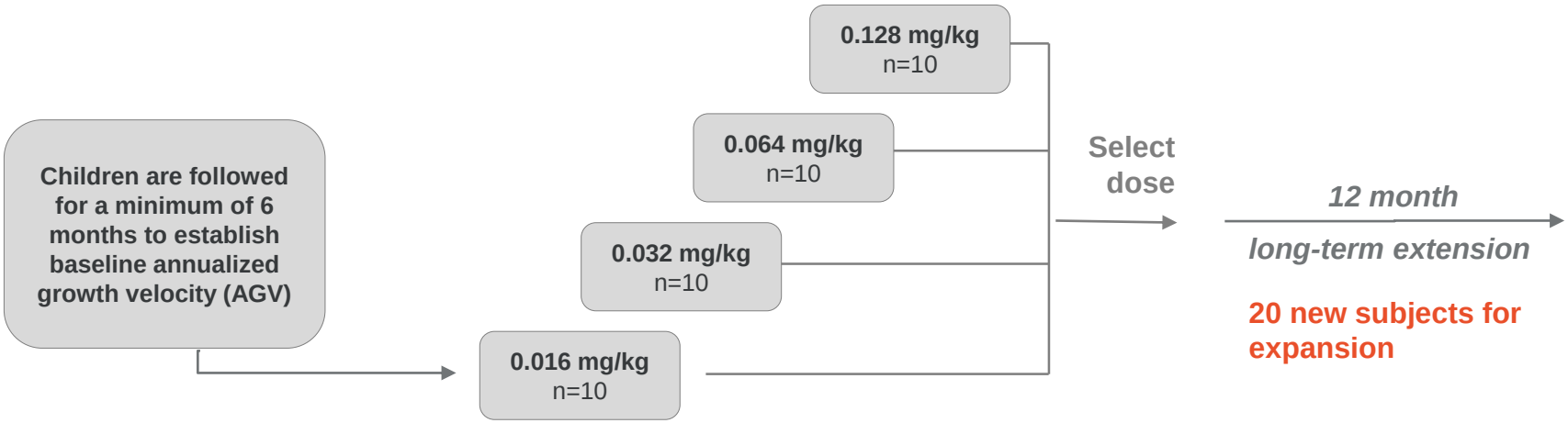
Percent increase compared to non-treated mouse

Company/ Asset	MOA	Route	Status	Mouse model	Tibia length	Femur length	Foramen magnum area	L4-L6 height
bridgebio Infigratinib	Selective FGFR1-3i	Oral	Ph2 lead-in ongoing[2]	$FGFR3^{Y367C/+}$	33%	21%	16%	12%
BIOMARIN Vosoritide (BMN111)	CNP analogue	Daily SQ	Pivotal	$FGFR3^{Y367C/+}$	7%	5%	No known publicly available data	3%
ascendis pharma TransCon CNP[1]	CNP analogue	Weekly SQ	Ph2 lead-in ongoing	$FGFR3^{Y367C/+}$	12%*			
Pfizer Reifercept (TA-46)	FGFR3 decoy	Weekly SQ	Ph1	$FGFR3^{ACH/+}$	9%*	7%*		

Source: Komla-Ebri et al. J Clin In2v 2016, Lorget et al. Am J Hum Genet 2012, Garcia et al. Science Trans Med 2013, Breinholt ENDO 2017
Note: subcutaneous doses, percent increase compared to vehicle treated $FGFR3^{Y367C/+}$, $FGFR3^{ACH/+}$ mouse as noted in "Mouse model" columns
Infigratinib treatment with 2mg/kg subcutaneous dose
[1]Based on vosoritide continuous infusion; *Value estimated using DigitizeIt. [2]Protocol submitted to Australian local ethics committed, IND submission to FDA expected 2020.

The PROPEL clinical program is enrolling and potential POC data expected in 2021

Observational run-in | **Ph2 Dose-finding (n=40)** | **Expansion (n=20)**



Key inclusion criteria
- Children 2.5 – 10 years old
- Clinical and molecular ACH diagnosis

Primary objectives
- Baseline annualized growth velocity (AGV)

Primary objectives
- Identify safe therapeutic dose for expansion / pivotal study
- Safety and tolerability
- Change from baseline in AGV

Primary objectives
- Long-term safety and efficacy

Recombinant collagen type VII for recessive dystrophic epidermolysis bullosa (RDEB)

Bardy, child with RDEB

RDEB overview:

- **Prevalence**: 1,500 (US + EU)
- **Genetic driver**: mutations in the COL7A1 gene encoding the protein collagen type VII
- **Pathophysiology**: Systemic impairment of dermal-epithelial cohesion throughout various tissues leading to painful blistering on the skin, GI tract, and oral cavity

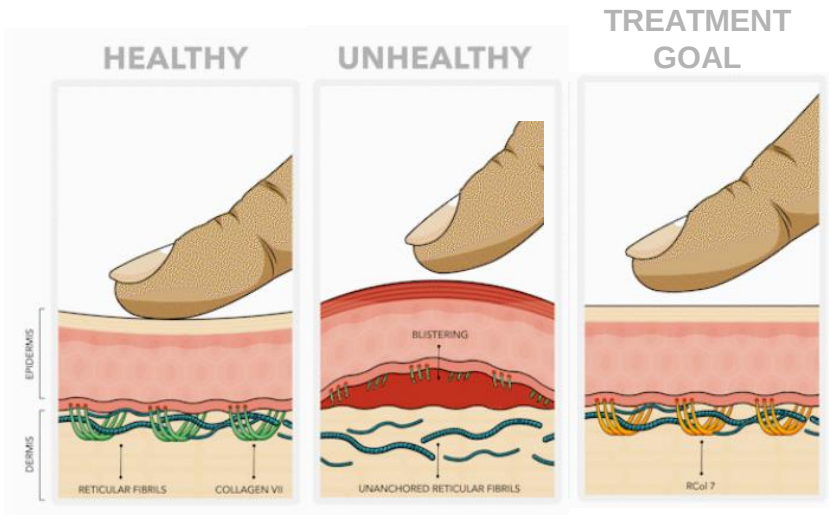
Features of a potential best-in-class medicine for RDEB:

- **Targeting RDEB at its genetic source,** by replacing missing COL7 protein via a simple IV infusion
- **Potential to address burden of RDEB beyond the skin**, including systemic manifestations
- **Proactively address wound formation and healing**, rather than reactively treat lesions

Recombinant collagen type VII for recessive dystrophic epidermolysis bullosa (RDEB)

RDEB COL7 loss-of-function mutations cause:

- Near complete loss of COL7 at epithelial junctions on the skin and throughout the body
- Painful erosions and blistering on the skin, GI tract, and oral cavity
- Failure to thrive, decreased life span, high risk for squamous cell carcinoma



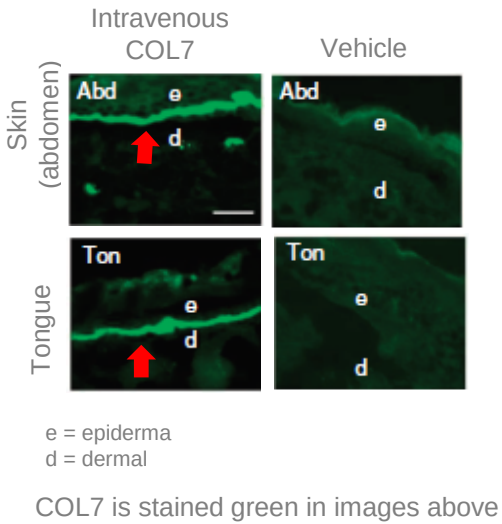
Our systemic COL7 replacement is designed to:

- Replace COL7 at epithelial junctions throughout the body
- Address the systemic burden of RDEB including on the skin, GI tract and oral cavity
- Proactively address wound formation and healing globally rather than reactively treat lesions

Recombinant COL7 distributes systemically, leading to survival benefits in the RDEB mouse model

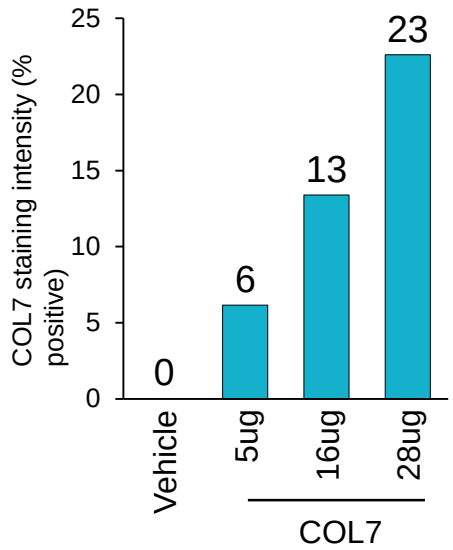
A single intravenous injection of recombinant COL7 distributed to epithelial barriers throughout the body (skin, oral cavity, GI tract), in a dose-dependent manner

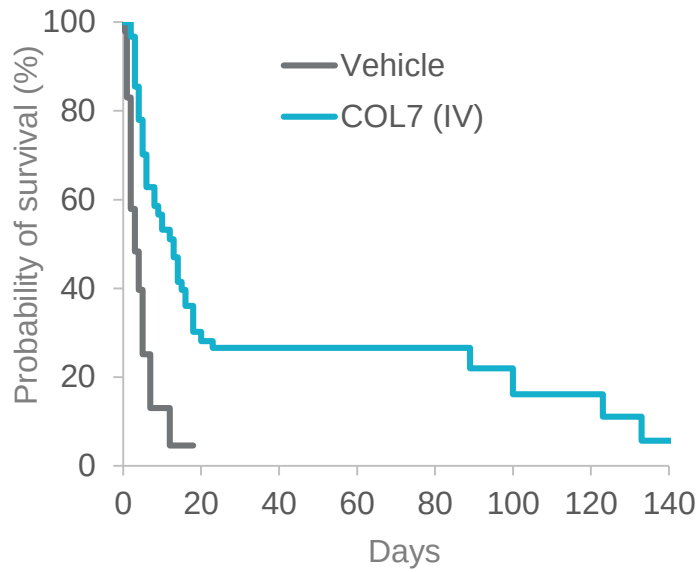
This led to a **significant survival benefit** in COL7-treated animals



e = epiderma
d = dermal

COL7 is stained green in images above



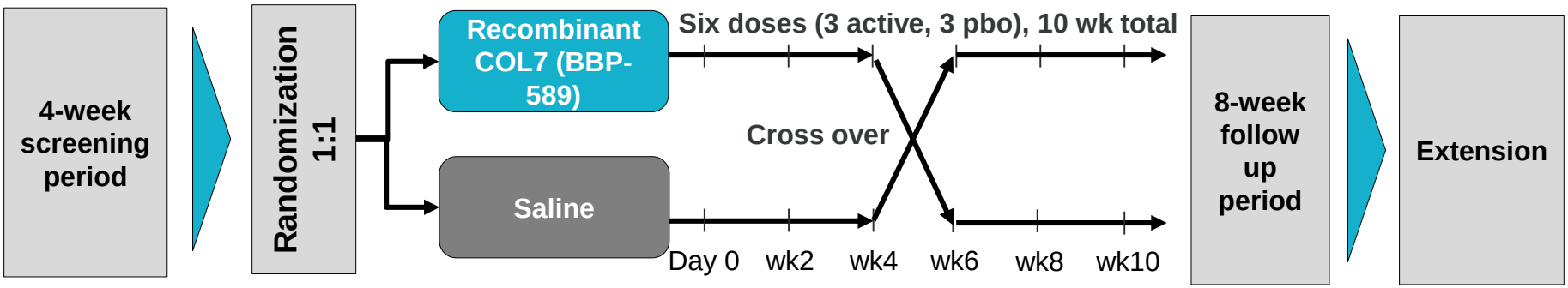


Source: Hou & Chen, *Journal of Investigative Dermatology* 2015

Ongoing randomized, dose-escalation Phase 1/2 proof-of-concept clinical study in adults with RDEB

- First patient dosed in 1Q19
- **Anticipate potential POC data in 2020**

Protocol for EACH cohort



Cohort 1: 0.1 mg/Kg N=2 → **Cohort 2: 0.3 mg/Kg N=4** → **Cohort 3: 1 mg/Kg N=6** → **Cohort 4: 3 mg/Kg N=4**

KEY INCLUSION CRITERIA

- Adult with RDEB diagnosis
- Deficiency but not total loss of COL7 protein
- At least 1 wound >20cm^2 for ≥6 weeks

KEY EXCLUSION CRITERIA

- Known hypersensitivity to BBP-589
- Received investigational RDEB agent in last 6 months

PRIMARY ENDPOINT

- Safety and tolerability

KEY SECONDARY AND EXPLORATORY ENDPOINTS

- COL7 deposition and residence time in skin biopsies
- Change in healing of up to 5 target wounds
- Patient reported outcomes (itch, QoL)

Targeted oncology portfolio

Andrea,
CCA patient

World-class oncology team drives our discovery and development

- **Eli Wallace:** CSO Oncology
- **Frank McCormick**, Chairman of Oncology
- **Richard Scheller**, Chairman of R&D

We target driver mutations in genetically defined cancers…

- **FGFR1-3i** for FGFR+ cancer: Near-term revenue in CCA, multiple expansion indications
- Pan-mutant **KRASi** for KRAS+ cancer: Platform approach in partnership with NCI RAS initiative

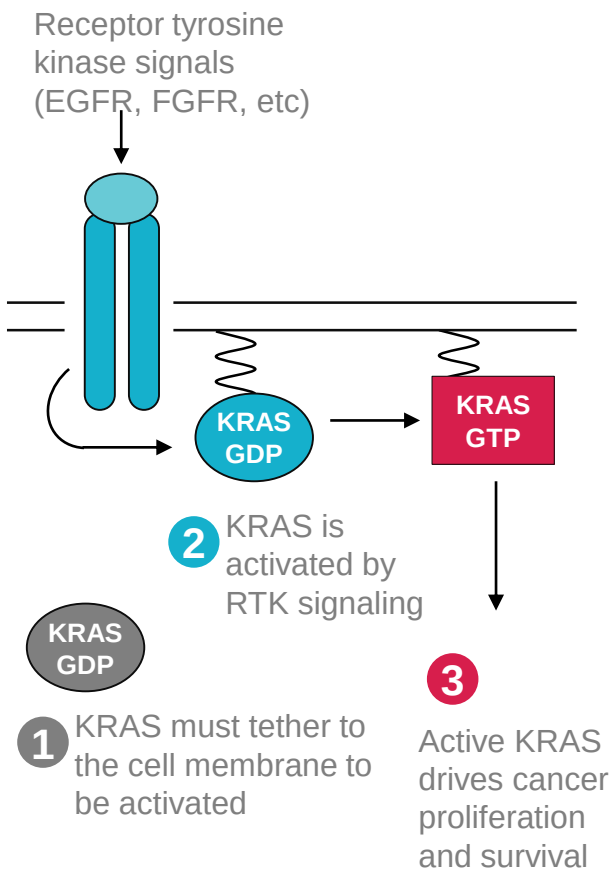
…while also focusing on novel targets with extensive academic validation

- **SHP2i** for multiple tumors (10+ recent papers in *Nature, Science, Nature Medicine*)
- **GPX4i** for multiple tumors (10+ recent papers in *Nature, Cell, Cancer Cell*)

Program	MOA	Disease	Stage	Next anticipated update
Infigratinib	FGFR1-3 inhibitor	FGFR+ cancer	Ph3	Pivotal CCA data 2020, NDA 2020
BBP-398	SHP2 inhibitor	Multiple tumor types	Pre-IND	IND submission in 2020
BBP-454	Pan-mutant KRAS inhibitor	KRAS+ cancer	Discovery	Clinical candidate nomination
BBP-954	GPX4 inhibitor	Multiple tumor types	Discovery	Clinical candidate nomination

KRAS: multiple shots on goal with our pan-mutant inhibitor programs – each with a unique MOA targeting a novel pocket

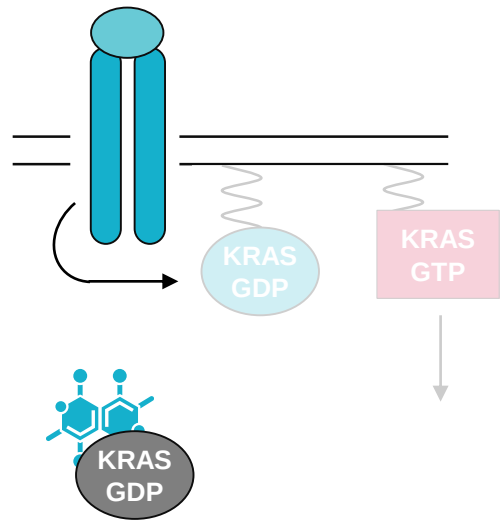
KRAS activation in cancer is a multi-step process



Our programs target different steps of the KRAS activation process

Program 1: C185 targeting

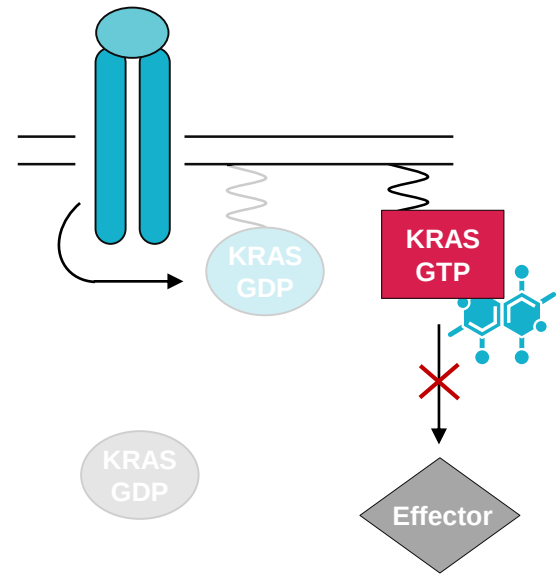
- Blocks KRAS from tethering
- Blocks conversion of inactive KRAS GDP to active KRAS GTP



KRAS tethering is blocked – cancer growth is inhibited

Program 2: H95 targeting

- Directly binds activated KRAS
- Inhibits KRAS from signaling through effectors



Activated KRAS signaling is inhibited

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SHP2: Our potentially best-in-class SHP2 inhibitor is expected to enter the clinic mid-2020

- SHP2 connects RTK signaling to downstream MAPK signaling activation
- Our compound potently traps SHP2 in an inactive state, thereby potentially blocking downstream oncogenic signaling
- In collaboration with MD Anderson, optimized our SHP2i for use in combination and reduced cardiac liability
  - No evidence of QTc prolongation or hypertension
- BBP-398 was well tolerated in rats and dogs in 28d GLP-tox studies
  - Histological and clinical chemistry findings consistent with MAPKi
  - At maximum doses (25 mg/kg/day, dogs; 100 mg/kg/day, rats), MTD was not reached
- IP published 02/13/2020
- First SHP2 inhibitor clinical data, (RVMD Q1 2020) demonstrates monotherapy antitumor activity*

Preclinical SHP2i data

In vitro properties	Navire	RVMD*
pERK IC_{50} (nM) cellular assay	<40	<40
hERG Patch clamp IC_{50} (μM)	>100	?
Monotherapy anti-tumor activity		
KRASG12C xenograft	✓	✓
EGFR mutant xenograft	✓	✓
Combination enhanced anti-tumor activity		
MEKi	✓ (trametinib)	✓ (cobimetinib)
EGFRi osimertinib	✓	✓

Preclinical profile demonstrates activity in-line with SHP2i class and potential for better tolerability

Source: *Revolution Medicines S-1

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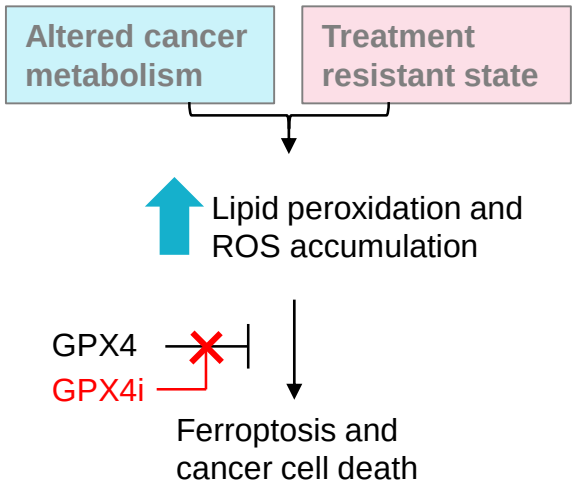
GPX4: Potential first-in-class therapy for a novel cancer target

MOA

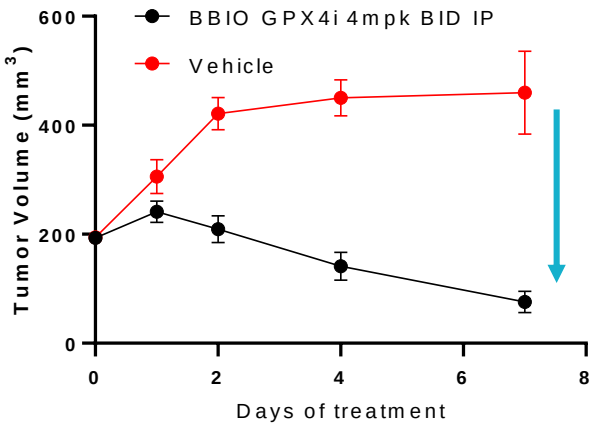


- GPX4 was recently identified as a key tumor dependency in multiple CRISPR screens and mechanistic studies
- GPX4 allows tumor cells to survive by neutralizing toxic lipid peroxides
- Our approach is to directly inhibit GPX4, thereby triggering cancer death through ferroptosis

Key data

In vivo monotherapy activity in a renal cell carcinoma mouse model

Model: 786-O RCC xenograft (VHL LOF, p53 LOF)



Rapid tumor regression after only 7 days of dosing

Synergy with targeted therapies and immunotherapy using in vitro models

Infigratinib (FGFRi): Near-term submission in CCA and multiple large expansion opportunities

Indication	Key Data	Status	Next planned update
FGFR2+ cholangiocarcinoma	**39% ORR** in patients with ≤1 previous line of treatment	▪ Enrollment complete in 2L Ph2 pivotal cohort ▪ Ph3 in 1L study enrolling	▪ Updated pivotal data 2H20 ▪ NDA submission 2H20 ▪ 2021 launch
FGFR3+ urothelial carcinoma	**25% ORR** in metastatic relapsed refractory setting suggests clear activity in this tumor	▪ FPI for Ph3 in adjuvant setting in 1H20	▪ Complete enrollment in Ph3 adjuvant study
FGFR fusion-positive tumor agnostic	**5 tumor types** Showed response to infigratinib in Ph1/2	▪ FPI for Ph2 signal optimization study in 1H20	▪ Potential Ph2 data 2021

SOURCE: Javle M et al. ESMO 2018, Pal S et al. Cancer Discovery 2018, data on file

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Gene therapy portfolio

Vayle, child with Canavan

Experienced team with track record in gene therapy

avexis · BIOMARIN · SAREPTA THERAPEUTICS · bluebirdbio · PRECISION BIOSCIENCES · NOVARTIS

Partnered with top academics in the gene therapy space

- Guangping Gao, Ph.D (UMass)
- Pierre Bougneres, M.D., Ph.D. (INSERM)
- Jeff Holt, Ph.D (Boston Children's)

Congenital adrenal hyperplasia (BBP-631)

- One of the largest known AAV gene therapy markets (prevalence 75K US+EU)
- Low threshold to correct phenotype, validated by human genetics
- Durable transgene delivery and expression for 6-month in NHP study

Canavan disease (BBP-812)

- Lethal, degenerative, neuromuscular disease
- Precedented AAV9 serotype with safety data in one compassionate use case

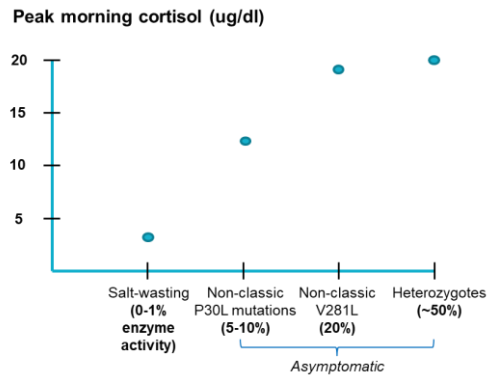
TMC1-driven hearing loss (BBP-815)

- Delivers functional copy of TMC1 gene allowing transmission of auditory stimuli
- Nature Communications publication shows significant rescue of hearing function in diseased mice

Program	MOA	Disease	Stage	Next anticipated update
BBP-631	21-OHase gene therapy	Congenital adrenal hyperplasia	Pre-IND	IND submission in 2020
BBP-812	ASPA gene therapy	Canavan disease	Pre-IND	IND submission in 2020
BBP-815	TMC1 gene therapy	Genetic hearing loss	Discovery	Clinical candidate nomination

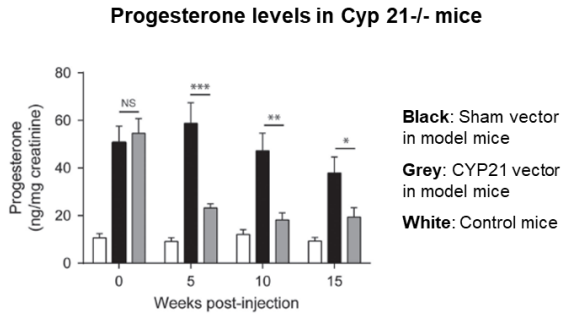
21-OH gene therapy for CAH: NHP study showed durable transgene expression; 5-10% of WT enzyme may be sufficient for clinical impact

Genotype-phenotype studies show that >5-10% of enzyme activity results in nonclassical CAH



- Due to the high enzymatic efficiency/selectivity of 21-OHase, **only a small amount of enzyme is required to rescue the phenotype**

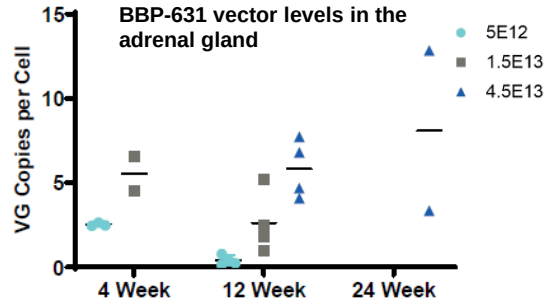
Mouse studies show a VGC of only 0.13 at 18 wks was sufficient for phenotypic correction



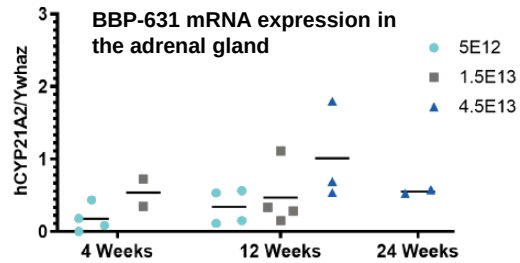
VGC from the whole adrenal gland was 0.13 at wk 18

- At 15 weeks in treated mice, **progesterone** (the key substrate of 21OHase in mice) was **significantly reduced vs untreated mice**

NHP studies show sustained VGC and RNA out to 6 months



- Mean vector genome copies per cell appear stable at 24 wks



- Transgene expression is dose-dependent and stable out at 24 wks

Source: ESGCT 2019

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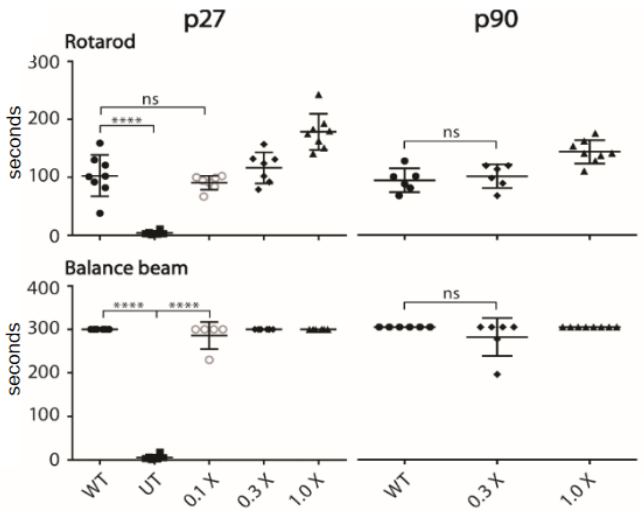
ASPA gene therapy for Canavan: Phenotypic correction in a lethal mouse model and broad CNS transduction in NHPs

Mouse studies show that BBP-812 can achieve phenotypic reversal

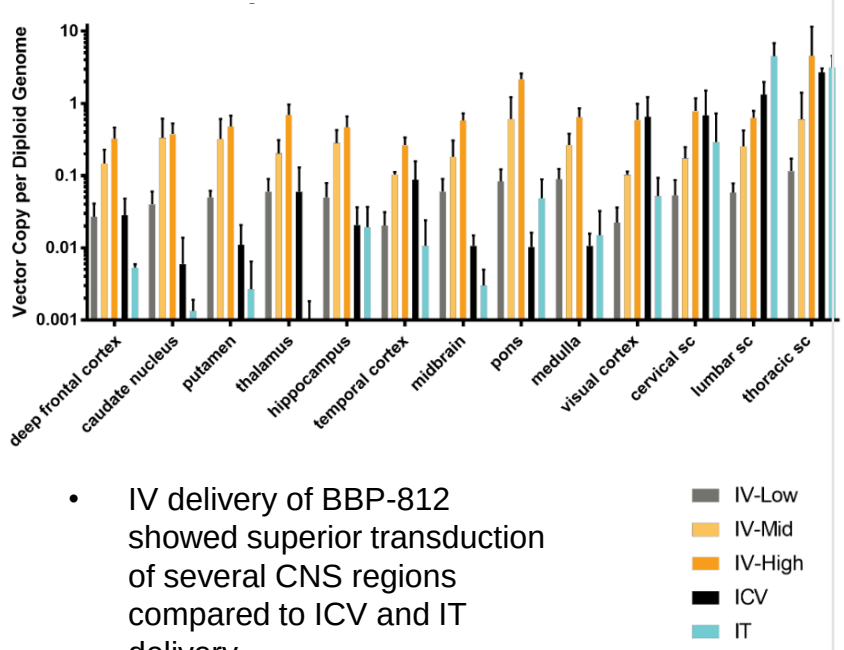
Effect of BBP-812 on rotarod and balance beam, ASPA KO mice (untreated vs 3 different doses) and WT mice



- ASPA KO mice treated with at least 2.6e13 vg/kg had NAA metabolism and performance on motor function tests restored. Mice treated at 2.6e14 vg/kg outperformed WT mice.

NHP studies show broad CNS distribution with IV delivery

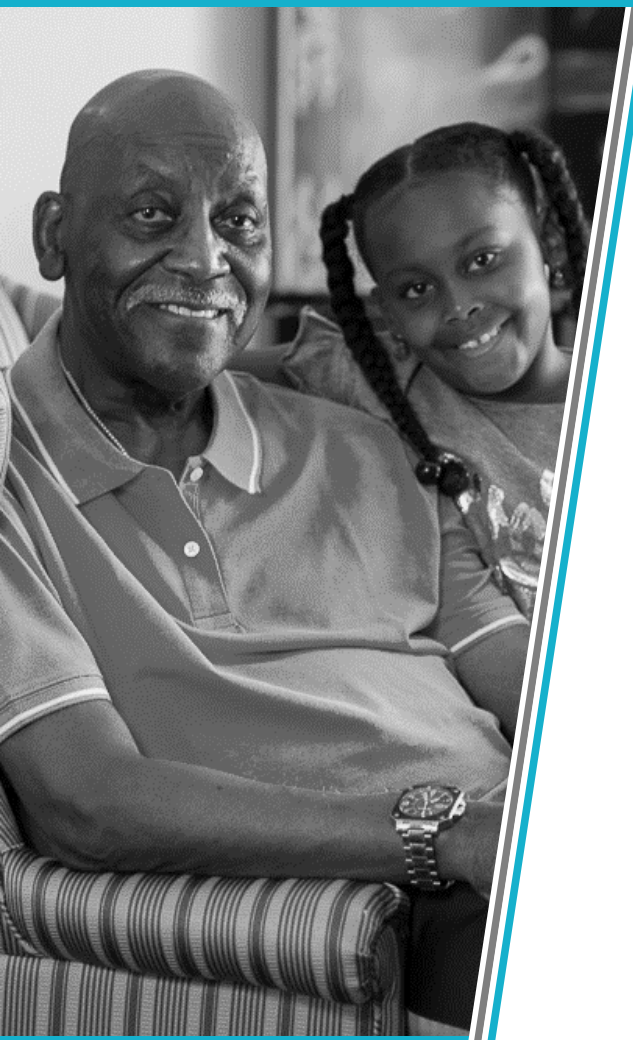
Vector copies per diplome in various CNS regions in NHPs



- IV delivery of BBP-812 showed superior transduction of several CNS regions compared to ICV and IT delivery

Source: ESGCT 2019

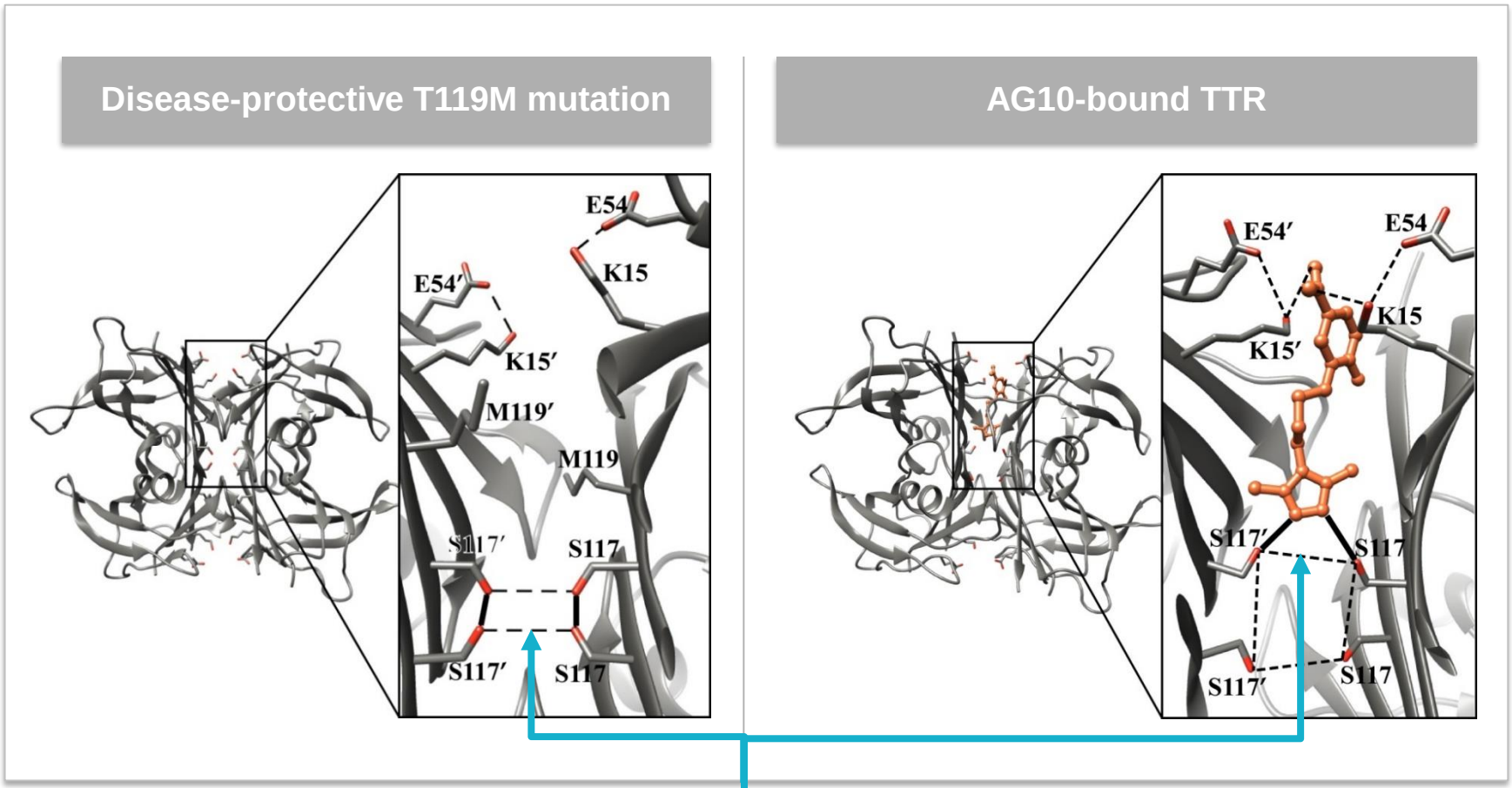
AG10 for TTR amyloidosis (Eidos)



Art, ATTR-CM patient

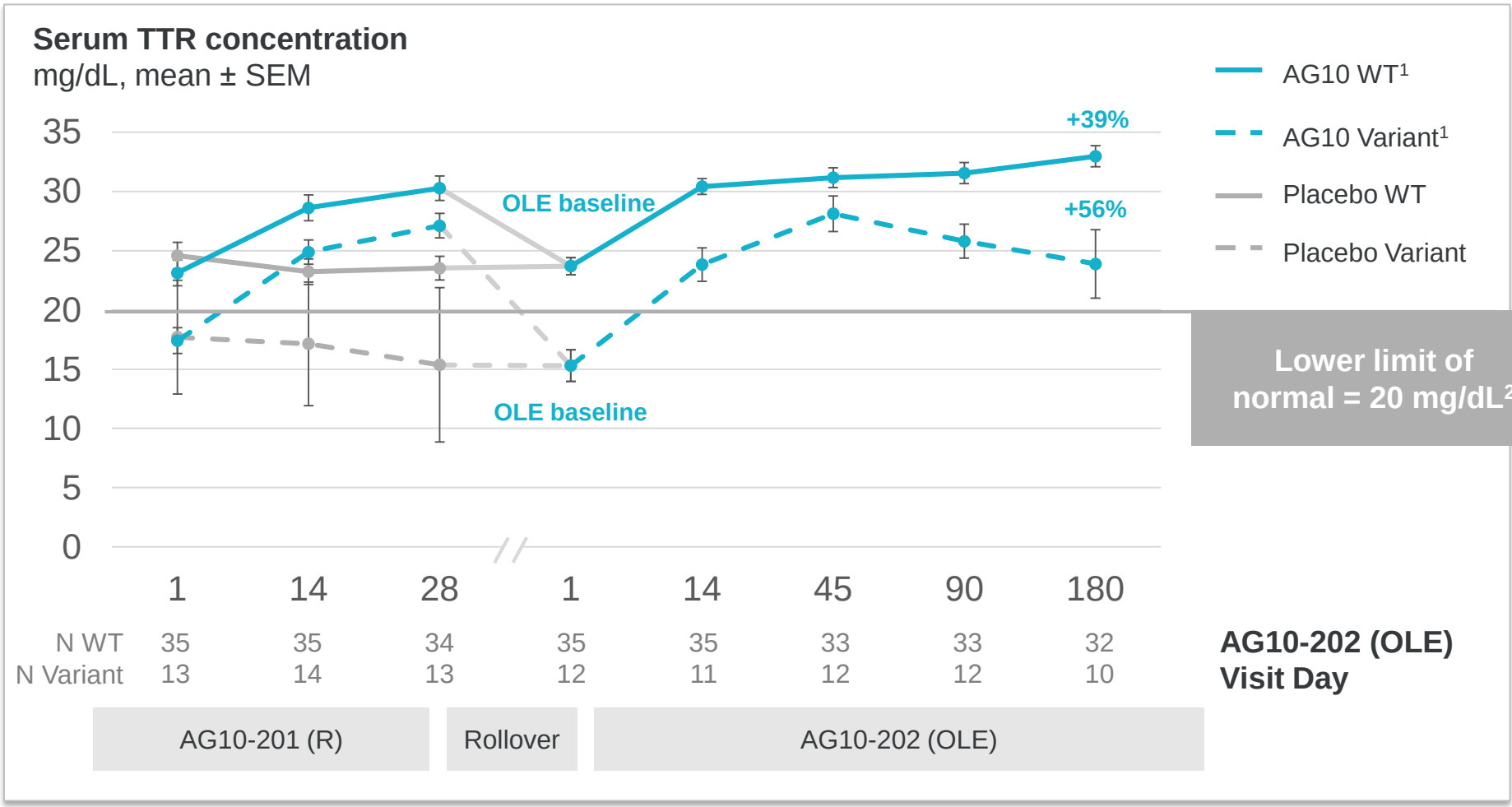
- **Addressing large and growing need in ATTR**, a fatal disease affecting >400K patients
- **Targeting the disease at its source** by stabilizing TTR, a genetic and clinically validated mechanism
- **Advancing AG10, a potential best-in-class drug** that mimics naturally occurring rescue mutation
- Phase 2 open label extension study suggests potential to **reduce mortality and cardiovascular hospitalizations** at 15 months
- **Executing Phase 3 study in ATTR-CM** with top-line data expected in 2021

AG10 structurally mimics disease-protective mutation by hyper-stabilizing TTR



Strong inter-monomer H-bonds observed via X-ray crystallography
Unique binding mode vs other stabilizers

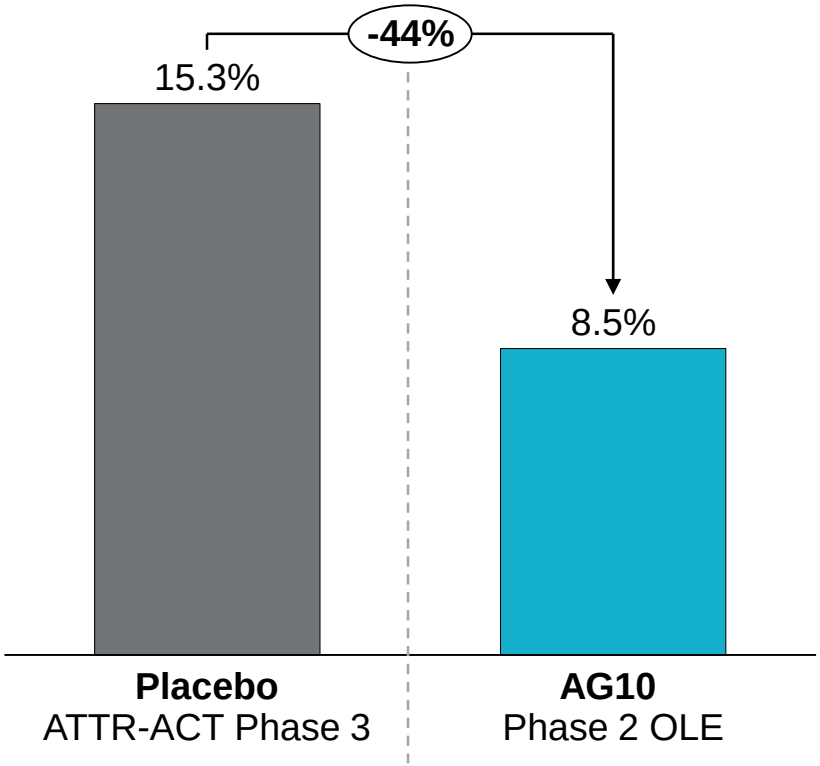
Serum TTR levels, a prognostic indicator of survival, increased upon AG10 treatment and were maintained throughout Ph 2 study



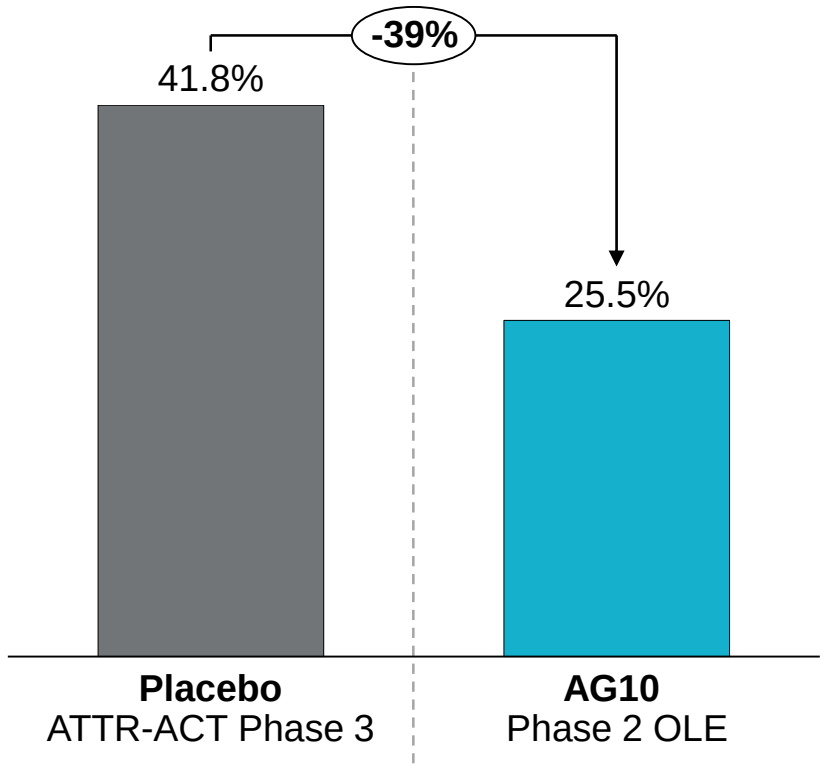
1 400mg and 800mg BID AG10 groups pooled during randomized portion
2 Defined as the lower limit of the reference interval for the serum prealbumin (TTR) clinical laboratory assay

Deaths and CV hospitalizations reported in AG10 Phase 2 OLE were lower than in placebo-treated ATTR-ACT participants

All-cause mortality at 15 months
Participants died or receiving transplant (%)



Cardiovascular hospitalizations at 15 months
Participants with ≥1 CV hospitalization (%)



Phase 3 ATTRibute study expected to complete enrollment in 2H20

1 Based on routine adverse event reporting
Note: These data are based on a cross-trial comparison and not a randomized clinical trial. As a result, the values shown may not be directly comparable

Fosdenopterin (cPMP replacement) for MoCD type A

Elliott, child with MoCD type A

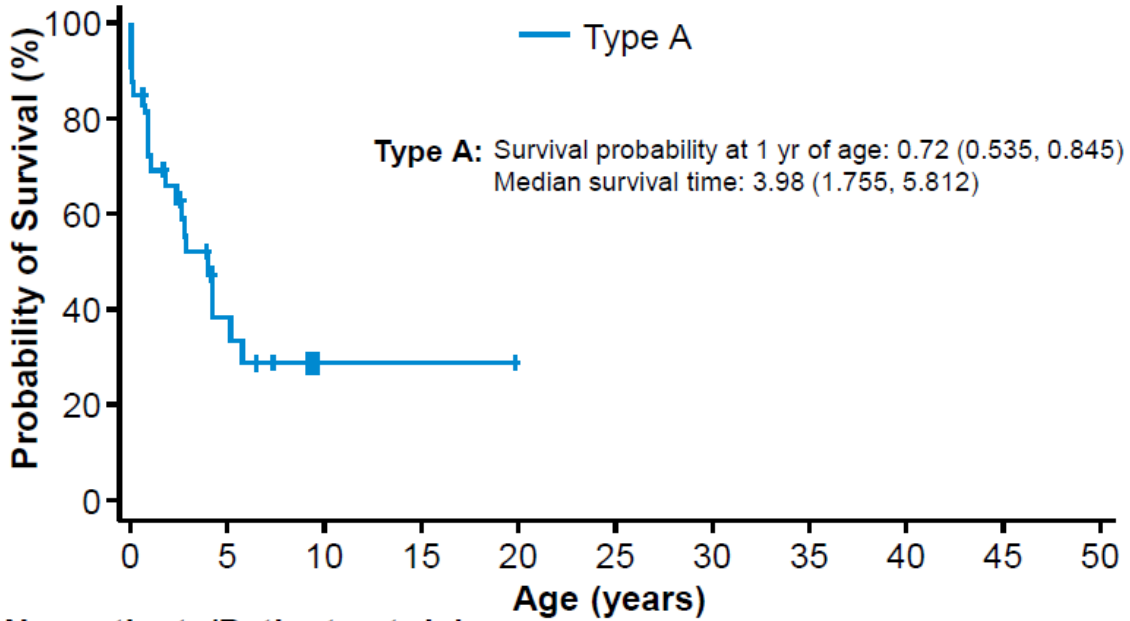
Genetic driver: MOCS1 / cPMP depletion
Prevalence (US + EU): 100

- **Designed to address an extreme unmet medical need in molybdenum cofactor deficiency (MoCD) type A**, a progressive and rapidly fatal CNS disorder (median survival < 4 years)
- **Targeting the disease at its source** by directly replacing cPMP, the missing metabolite that causes CNS toxicity
- **Potentially life saving investigational drug** with compelling pivotal data showing prolonged survival, seizure control and ambulation vs natural history
- **Rolling NDA submission initiated in 4Q19**, under FDA Breakthrough Therapy Designation

We presented data from our natural history study in MoCD type A at SSIEM 2019

- Median survival time of <4 years highlights urgent need for a new medicine
- Data will play an important role in our NDA data package

Kaplan-Meier Estimates of Survival Probability (FAS)



Type A: Survival probability at 1 yr of age: 0.72 (0.535, 0.845)
Median survival time: 3.98 (1.755, 5.812)

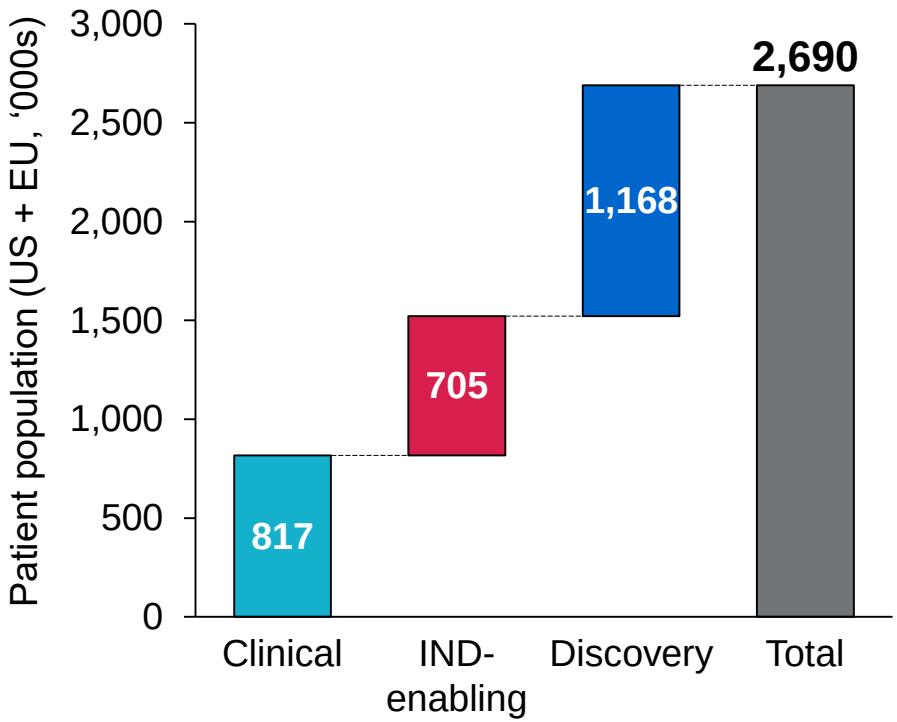
No. patients/Patients at risk

Type A:	0/33	18/8	20/1	20/1	20/0	20/0	20/0	20/0	20/0	20/0	20/0

Note: Kaplan-Meier curves step down at time points at which a death has been observed; slashes represent patients whose observation time was censored as of the last contact date.

Our current pipeline has the potential to treat nearly 3 million patients in the US and EU alone

Patient population by development stage



Breakdown of clinical-stage assets

Indication	Population
ATTR	400,000
Hypoparathyroidism	200,000
Basal cell carcinoma	120,000
Achondroplasia	55,000
FGFR+ cancer	37,000
Inherited retinal dystrophy	3,000
RDEB	1,500
MoCD type A	100
Total:	**817,000**

Our product platform has the potential to deliver diversified and sustainable revenue growth beginning in 2021

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Assessing BridgeBio

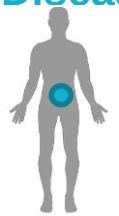
	Criteria	Relevance	Focus Today
1	High probability of success	• Historically higher probability of success for genetic disease drugs • BridgeBio's early programs have outperformed historical probabilities	Current Pipeline Progress
2	Number of programs	• We find great science and unlock its potential for patients • Always searching for the next PellePharm or Eidos • Scale allows for objective assessment and failure	New Programs
3	Capital efficiency	• Generate value by making each program ROI-positive • Driven by judicious use of capital at the high-risk preclinical stages	Spend to IND

We recently announced four new programs including two entering Phase 2 trials

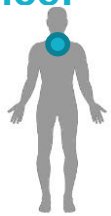
Encaleret

Mechanism: Ca sensing receptor antagonist

Diseases and prevalence:



Autosomal dominant hypocalcemia type 1
2,000
US + EU



Hypoparathyroidism
200,000
US + EU

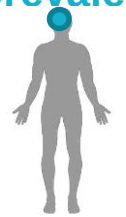
Modality: Small molecule

Phase 2 ready

Zuretinol

Mechanism: Synthetic retinoid

Diseases and prevalence:



Inherited retinal disease caused by RPE65 or LRAT mutations
3,000
US + EU

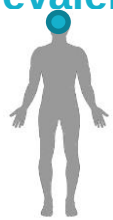
Modality: Small molecule

Phase 2 ready

BBP-472

Mechanism: PI3Kβ inhibitor

Diseases and prevalence:



***PTEN* autism**
120,000
US + EU

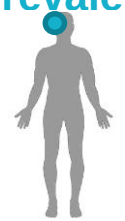
Modality: Small molecule

Discovery

BBP-815

Mechanism: TMC1 gene therapy

Diseases and prevalence:



Genetic hearing loss
10,000
US + EU

Modality: Gene therapy

Discovery

We plan to announce multiple additional new programs in 2020

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Encaleret (CaSR antagonist) for hypoparathyroidism

Encaleret targets disease at its source by selectively antagonizing the CaSR, a key regulator of calcium homeostasis

- Opportunity to develop encaleret was identified in collaboration with global experts at the NIH
- Being prosecuted by the BridgeBio cardiorenal group

Encaleret is a potential 1st in class CaSR antagonist with differentiated profile for hypoparathyroidism

- Initial genetically-defined population of autosomal dominant hypocalcemia type 1 (ADH1), provides high probability of success
- Potential for expansion into broader hypoparathyroidism indication (~200K patients in US & EU)

Prior clinical experience with encaleret enables accelerated clinical development

- Well tolerated in >1,200 human subjects and increased serum calcium in a dose-dependent manner
- IND application submitted in late 2019, currently Phase 2 ready
- Proof-of-concept data in ADH1 expected in 2021

Assessing BridgeBio

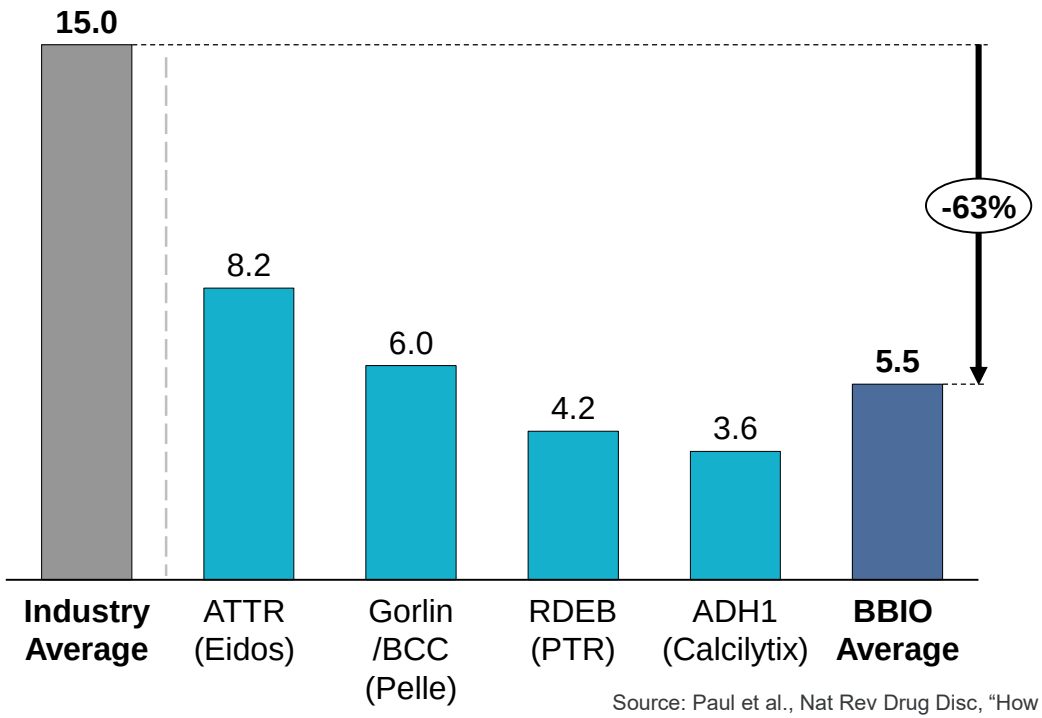
	Criteria	Relevance	Focus Today
1	High probability of success	• Historically higher probability of success for genetic disease drugs • BridgeBio's early programs have outperformed historical probabilities	Current Pipeline Progress
2	Number of programs	• We find great science and unlock its potential for patients • Always searching for the next PellePharm or Eidos • Scale allows for objective assessment and failure	New Programs
3	Capital efficiency	• Generate value by making each program ROI-positive • Driven by judicious use of capital at the high-risk preclinical stages	Spend to IND

On average, we have brought assets forward more efficiently than industry average

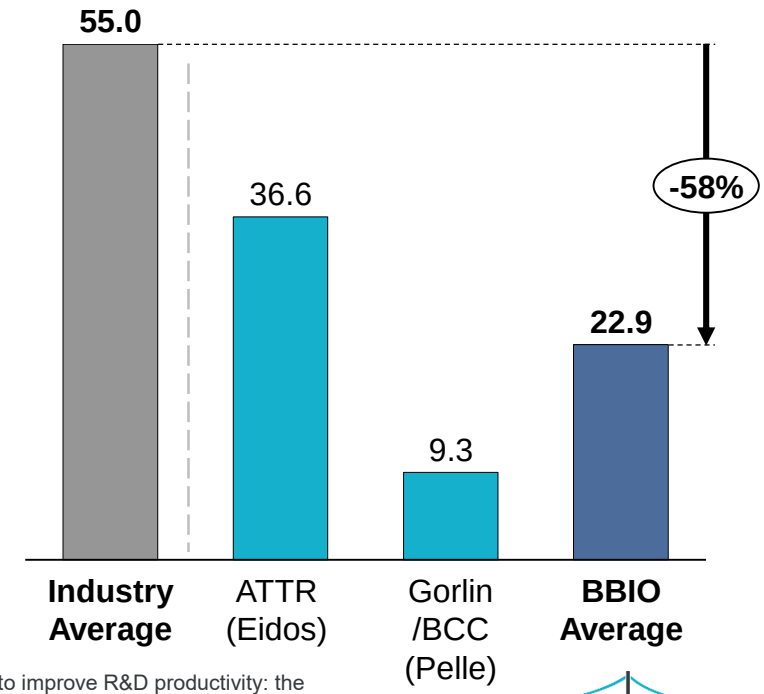
Operationally Efficient Platform

- Our track record to date **is ~$6mm to IND** and **is ~$23mm IND to POC (Phase 2)**
- We aim to **rapidly and decisively** advance our product candidates to objective critical decision points
- We field a minimum viable team for each asset, with the goal of ensuring that each program has sufficient personnel to fit its purpose while **reducing excess overhead cost**

Spend to IND ($mm)



Spend IND to POC ($mm)



Note: BBIO values exclude license and acquisition costs.

Source: Paul et al., Nat Rev Drug Disc, "How to improve R&D productivity: the pharmaceutical industry's grand challenge.", 2010

2019 included a range of accomplishments across our development programs and operations

Clinical, regulatory, and scientific

- ✓ **ATTR:** initiated Ph3 trial, presented Ph2 open-label extension data
- ✓ **Achondroplasia:** initiated Ph2 observational lead-in, established therapeutic window between human safety database and projected efficacious achondroplasia doses
- ✓ **RDEB:** initiated Ph2 POC trial
- ✓ **MOCD Type A:** initiated rolling NDA
- ✓ **BCC:** completed Gorlin Ph3 enrollment; initiated Ph2 high frequency BCC
- ✓ **Oncology:** SHP2 combination data w/ MEK, EGFR, KRAS augmenting inhibition; GPX4 demonstrated monotherapy activity in mouse model
- ✓ **CCA:** fast track designation for first line, completed enrollment in second line efficacy cohort
- ✓ **CAH:** demonstrated 6-month durability in adrenal cortex
- ✓ **Canavan:** demonstrated broad CNS distribution using IV route of administration

Operations and finance

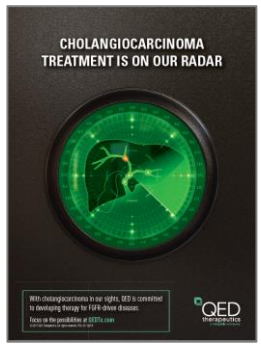
- ✓ **Building commercial organization:** Jennifer Cook, BOD member and commercial advisor; appointed Matt Outten as CCO
- ✓ **Financing:** raised over $650M in IPO and private financing

BridgeBio: Commercial build out

Top talent makes a difference

- **CCO: Matt Outten**
  - 20+ years pharma/biotech
  - Multiple commercial leadership positions across sales, marketing, market access
  - Led the successful launch of Imbruvica for 6 indications
- **25 BBIO leadership roles:**
  - VPs of Marketing, Market Access, Distribution, Commercial Operations, Directors of Marketing and Training, Data Analytics and Operations
  - In-field teams established: Clinical Trial Liaisons, Professional Services

Building awareness



Gearing up for genetic disease launches

- Critical **patient identification capabilities expanded** in rare diseases with multiple data sources
- Allows better target planning, asset review, and appropriate resource allocation
- Developing **best in class HUB and patient assistance programs** in prep for commercial launches
- **Developing tailored launch plans** for each sub, from brand development to promotional material and in-field team training

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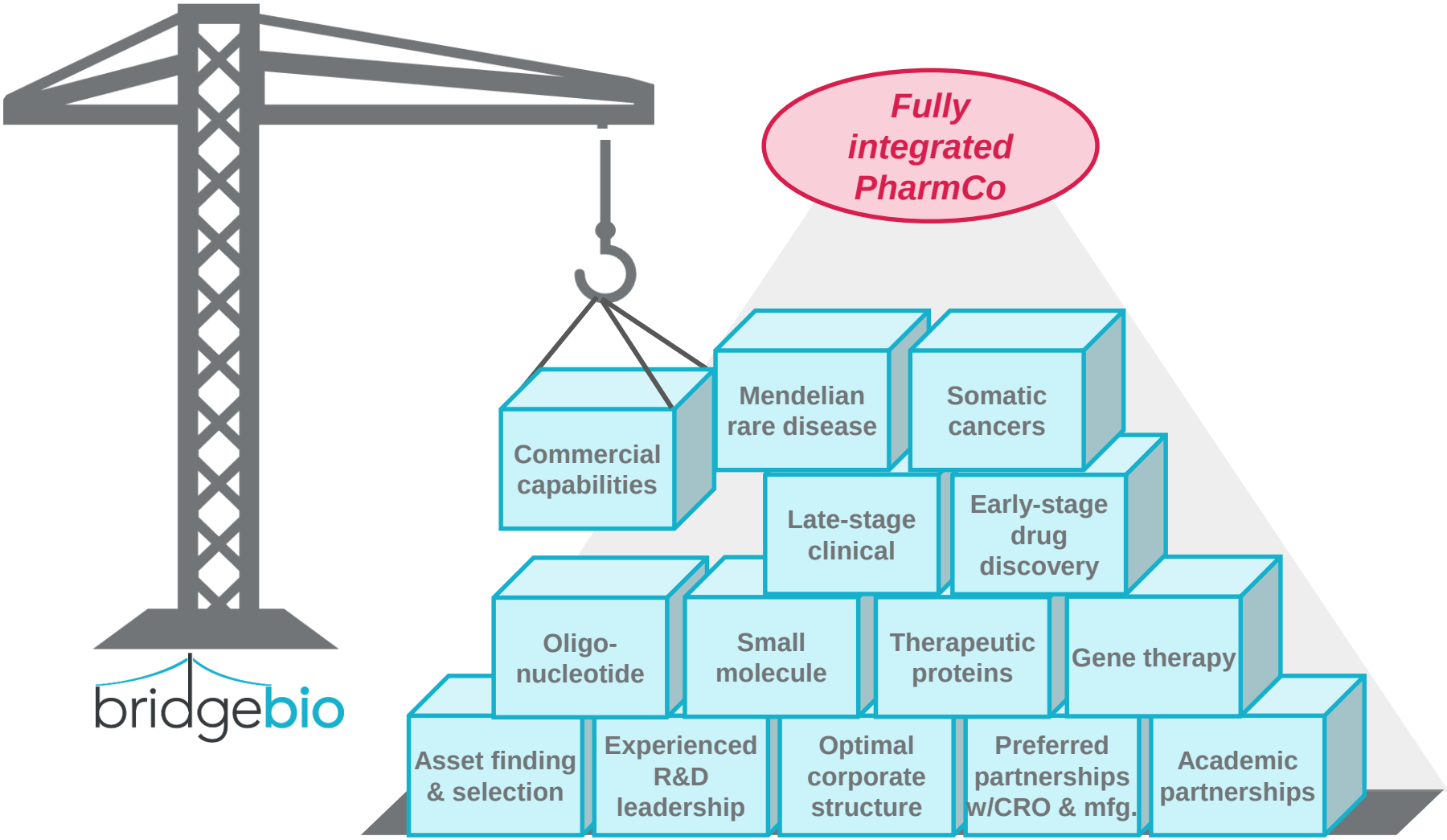
We are building a leading integrated pharma company



Multiple catalysts anticipated in 2020-2021

ESTIMATED

2020

1H

- ✓ New program announcements
- ❑ FGFRi for cancer: FPI Ph3 adjuvant urothelial carcinoma study
- ❑ FGFRi for cancer: FPI Ph2 FGFR fusion tumor agnostic Ph2 study

2H

- ❑ **Recombinant COL7 for RDEB: Topline Ph1/2 data**
- ❑ FGFRi for cancer: Pivotal 2L CCA data
- ❑ **Low-dose FGFRi for achondroplasia: Begin dosing Ph2**
- ❑ **TTR stabilizer for ATTR: Complete enrollment of ATTR-CM Ph3**
- ❑ FGFRi for cancer: Submit NDA for 2L CCA
- ❑ cPMP for MoCD type A: Complete NDA submission
- ❑ Multiple new IND filings

2021

FY

- ❑ **TTR stabilizer for ATTR: Topline data Ph3 Part A in ATTR-CM**
- ❑ **Topical SMOi for Gorlin: Topline Ph3 data**
- ❑ **Low-dose FGFRi for achondroplasia: Ph2 PoC data**
- ❑ **CAH gene therapy: Ph1/2 PoC data**
- ❑ **FGFRi for cancer: 2L CCA approval and launch**
- ❑ **cPMP for MoCD type A: Approval and launch**
- ❑ **CaSR antagonist for ADH1: Ph2 POC data**

Strong balance sheet expected to provide runway into 2022:
$577mn as of YE19 plus $550mn in gross proceeds from recent convertible debt offering

bridgebio